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EPIDEMIC OF FEBRILE DISEASE IN BERBERA, SOMALIA

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In April 1989 an epidemic of febrile illness occurred in Berbera, northern Somalia, several weeks after heavy rainfall, flooding and increase in mosquito populations. A malaria epidemic was ongoing in nearby Djibouti. Malnutrition and diarrhoea were endemic. Symptoms were influenza-like, lasted approximately 1 month and progressed to shock and death in about 700 persons between April 4- June 20, 1989. A WHO team detected falciparum malaria among 16/25 patients, all negative by Widal tests. In July 1989 a U.S. Navy team administered a questionnaire and obtained blood samples from 100 outpatients at the District Hospital. Peripheral smears were positive for malaria in 16/82 patients (13 falciparum). An additional patient, still symptomatic, had confirmed falciparum malaria which resolved with treatment (fansidar). IgM antibody to dengue virus was detected in only 3%. IgG antibodies were detected for Dengue virus in 59% West Nile Fever virus in 34%, Yellow Fever virus in 30%, Rift Valley Fever and Congo-Crimean Haemorrhagic Fever viruses in 7% each, Chickungunya virus in 4%, Ebola and Marburg viruses in 1% each, Rickettsia conorii and Coxiella burnetii in 40% each and Rickettsia typhi in 13%. The agent responsible for the epidemic could not be confirmed, but serological evidence of arboviral and rickettsial infections requires that these agents be considered in future epidemics.

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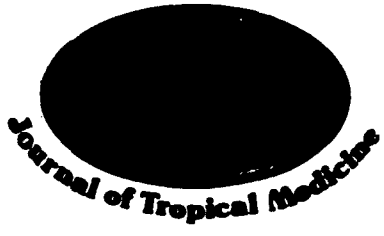

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EPIDEMIC OF FEBRILE DISEASE IN BERBERA, SOMALIA

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Abstract

In April 1989 an epidemic of febrile illness occurred in Berbera, northern Somalia, several weeks after heavy rainfall, flooding and increase in mosquito populations. A malaria epidemic was ongoing in nearby Djibouti. Malnutrition and diarrhoea were endemic. Symptoms were influenza-like, lasted approximately 1 month and progressed to shock and death in about 700 persons between April 4-June 20, 1989. A WHO team detected falciparum malaria among 16/25 patients, all negative by Widal tests. In July 1989, a U.S. Navy team administered a questionnaire and obtained blood samples from 100 outpatients at the District Hospital. Peripheral smears were positive for malaria in 16/82 patients (13 falciparum). An additional patient, still symptomatic, had confirmed falciparum malaria which resolved with treatment (fansidar). IgM antibody to dengue virus was detected in only 3%. IgG antibodies were detected for Dengue virus in 59%, West Nile Fever virus in 34%, Yellow Fever virus in 30%, Rift Valley Fever and Congo-Crimean Haemorrhagic Fever viruses in 7% each, Chickungunya virus in 4%, Ebola and Marburg viruses in 1% each, *Rickettsia conorii* and *Coxiella burnetii* in 40% each and *Rickettsia typhi* in 13%.

The agent responsible for the epidemic could not be confirmed, but serological evidence of arboviral and rickettsial infections requires that these agents be considered in future epidemics.

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Introduction

Berbera, Somalia, situated on the Gulf of Aden, adjacent to the Indian Ocean, is a coastal town approximately 260 Km. east of Djibouti on the Horn of Africa. This rocky, barren region of approximately 60,000 inhabitants occupies the northern-most part of Somalia and is one of the main ports of Somalia, supporting little agricultural activity except for animal herding. The hot, dry and windy summers force an annual migration of all but approximately 10,000 inhabitants to more elevated, cooler mountainous areas. Basic services in Berbera that include potable water and health care are provided nominally by the Ministry of Health (MOH), the Somali Red Crescent and other international and private voluntary agencies. However, prolonged drought conditions and civil war resulted in sizeable refugee populations from Ethiopia and other parts of Somalia, placing additional demands on medical care

resources that are at best insufficient. Disease problems were further aggravated by the poor nutritional status observed among the resident and refugee populations, with marasmus and kwashiorkor commonly reported by Somali Red Crescent volunteers.

In the period prior to and during the outbreak of febrile illness, heavy rains and flash flooding resulted in 1) the disruption of the fresh water pipeline from the mountains; 2) the contamination of water supply in deep wells; and, 3) breeding pools that resulted in a notable increase in vector arthropod populations (communications with Somali Red Crescent).

An epidemic of febrile illness began in April 1989 and was reported to have affected up to 50% of some population areas. The illness was characterized by fever and chills, headache, myalgias, arthralgias, vomiting and excessive sweating. Much less common were diarrhoea and jaundice. The disease lasted up to 6 weeks, apparently complicated by malnutrition, dehydration and anemia and resulted in shock and death usually within 1 week, or slow recovery.

Results of previous investigations revealed 60-78% positive slide tests for falciparum malaria, negative Widal tests (25) for typhoid fever, a reportedly positive response to treatment of over 3000 patients with anti-malarials, tetracycline, food and supportive treatment, and an estimated death toll of 700 (10-15 deaths per day) among the approximately 10,000 residents during the two months of April and May 1989 (MOH and WHO Reports, unpublished). At the request of the Somali Ministry of Health, a U.S. Navy team conducted a retrospective epidemiological study in July 1989, in order to obtain information on the etiology of this outbreak.

Materials and Methods

With the cooperation of the District Medical Officer, the Somali Ministry of Health (MOH) and the Somali Red Crescent, the investigation team was allowed access only to volunteers who attended the Berbera Hospital outpatient clinic. Questionnaire data and a blood sample were obtained from 100 available volunteers (75 males and 25 females) and both thick and thin peripheral smears were prepared to determine malaria infection. Sera were screened for evidence of arboviral and/or rickettsial infection, including tests for IgG class antibodies to Ebola virus, Marburg virus, Chikungunya virus, Rift Valley Fever virus, Crimean-Congo Haemorrhagic Fever virus, Dengue virus, West Nile (WN) Fever virus and Yellow Fever (YF) virus using ELISA, and *Rickettsia typhi*, *Rickettsia conorii* and *Coxiella burnetii* IgG antibody by ELISA (Williams et al., 1986). Sera were also tested for IgM class antibody to dengue, WN and YF viruses by ELISA (Voller et al., 1976). Personal data were collected from these volunteers using a standardized questionnaire.

Results

The mean age of the study population was 33.75 years, ranging from <1 to 82 years. Somalia was the reported birthplace of 83% of the respondents and Ethiopia by 15%. Fifty-one percent of the study population were permanent residents of Berbera, 37% temporary residents (assigned to this area by private voluntary relief organizations or the Ministry of Health) and 12% were displaced persons. Sixty-three percent (37/59) were unemployed and only 16% (11/59) were engaged in agricultural practices or animal herding. Most subjects reported illness during the period between April-May 1989. Non-specific, influenza-like symptoms predominated (headache, back pain, myalgias, constipation, chills, etc.) although only 31% reported fever. Few reported jaundice or enteric symptoms.

Most (61%) sought medical attention.

Evidence of IgG antibody was detected in sera tested for Dengue virus (59%), West Nile Fever virus (34%), Yellow Fever virus (30%), Crimean-Congo Haemorrhagic Fever virus (7%), Rift Valley Fever virus (7%), Chikungunya virus (4%), Marburg virus (1%), Ebola virus (1%), Rickettsia conorii (40%), Coxiella burnetii (39%) and Rickettsia typhi (13%). In addition, IgM antibody to Dengue virus (3%), WN (4%) and YF (6%) was suggestive of recent flaviviral infection. Prevalence figures for selected viral and rickettsial specific infections by age are provided in table-1. Previous multiple infections, or cross reactivity, were found in most (61%) sera tested for the viruses and rickettsiae. Conversely, only 17% of the sera tested were negative for antibody to one or more of the agents. Of 82 slides available for thick and thin smear examination, 16 (20%) were positive for malaria (13 falciparum and 3 vivax) and age-specific prevalence of malaria infections is noted in table-2. An additional patient (16 year old female), who had been bedridden since April (3 months) and had been treated unsuccessfully with oral and IM chloroguine and iron, was diagnosed as having falciparum malaria (presumably chloroquine-resistant) and was successfully treated in a Mogadishu hospital with fansidar (3 tablets orally) and a blood transfusion.

Table- 1: Age-specific prevalence of selected arboviral and rickettsial IgG antibody, Berbera, Somalia, July 1989.

Age	Total No.	*Rickettsia conorii*	*Coxiella burnetii*	Dengue Fever	West Nile Fever	Yellow Fever
0-9	2		50%			
10-19	9	66%	33%	55%	11%	11%
20-29	38	34%	40%	55%	26%	24%
30-39	16	63%	50%	63%	38%	31%
40-49	11	1%	45%	73%	55%	5%
50-59	10	30%	30%	60%	50%	60%
≥ 60	10	50%	30%	70%	40%	30%
Total	96	40% *(29-50%)	40% (29-50%)	59% (50-69%)	34% (25-45%)	30% (21-40%)

* (95% confidence interval)

Table- 2: Age-specific prevalence of malaria infections Berbera, Somalia, July 1989.

Age	Total No.	*Plasmodium falciparum*	*Plasmodium vivax*
0-9	5		
10-19	9	11%	
20-29	26	27%	4%
30-39	15	7%	
40-49	6	17%	33%
50-59	7	14%	
60-69	4	25%	
70-79	1		
80-89	2		
unknown	7	14%	
Total	82	16% *(9-25%)	3% (<1-10%)

* (95% confidence interval)

Discussion

Political upheaval created an environment of refugee populations, lack of food and an inadequate health care system under normally drought conditions. This, added to the heavy rainfall, which caused flooding, a reported increase in mosquito populations and disruption of drinking water supplies, were all critically interlinked factors contributing to the potential for epidemic disease, such as malaria or typhoid fever, complicated by malnutrition and dehydration. While little is known of the etiology of febrile illness in human populations in Somalia, data indicate that Dengue type 2 virus was responsible for disease outbreaks in 1985, 1986, and 1987 near Hargeysa, Somalia, characterized by up to 10 days of fever, chills, sweats, headache, back and joint pains (Botros et al., 1989). Rises in dengue virus IgM antibody titer were noted among expatriates in Somalia in 1983, although a survey conducted the following year yielded no evidence of infection (Salch et al., 1985). High prevalence of IgG rickettsial antibodies has been documented in North Africa (Corwin et al. 1992). However, infection has yet to be associated with human disease. Vector survey studies in Somalia have identified a number of mosquito species capable of transmitting malaria, including *Anopheles d'thali*, widespread in the northern region, and *Anopheles gambiae* and *Anopheles funestus* throughout the country. Malaria outbreaks were documented in human populations in 1981-82, and temporally linked with a period of heavy rainfall (Zahar, 1984). *Plasmodium falciparum* was found to be the cause of febrile illness among humans in Dijibouti (Fox et al., 1989), which shares a common border with Somalia in close proximity to Berbera.

Malaria is reportedly endemic in Berbera (WHO and MOH reports, unpublished), as well as in nearby Djibouti (Fox et al., 1989) and Ethiopia (Schubert et al., 1983 and Teklehaimanot,

1986). Chloroquine resistance has been reported in Somalia (Warsame et al., 1988) and areas of Ethiopia (Teklehaimanot, 1986) bordering Somalia, and unofficially reported (French Army physician communication) in Djibouti. Interestingly, Djibouti, approximately 260 Km west of Berbera, experienced an outbreak of falciparum malaria associated with severe shock among Ethiopian refugees, 6 dying within 12-48 hours of admission during the first week of June, 1989 (WHO communication, unpublished).

During this investigation, a control population was not accessible, thus, findings were limited to descriptive rather than analytical analysis. The absence of more detailed information precluded temporal estimates of symptom onset and duration, and many symptoms may have been forgotten 3 months after illness. Serological evidence of previous exposure was based on a single serum specimen collected approximately 3 months after the acute phase of illness. Consequently, virus isolation was not attempted. Since dengue, West Nile and Yellow fever are antigenically closely related, these data (table-1) can only be interpreted as evidence of flaviviral infection.

In summary, findings do not allow for definitive identification of the etiological agent or agents responsible for the outbreak. Data were acquired under extremely adverse environmental and political conditions, as currently occur in Somalia and other regions of the world, and which adversely impact on health. Nevertheless, these data provide evidence that faciparum malaria, complicated by malnutrition and dehydration, was a possible cause of this epidemic, and that many infectious agents such as arboviruses and rickettsiae are also endemic in this coastal area of Somalia and thus must be considered in future epidemics.

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